DOCUMENT RESUME

ED 205 872 CG 015 364

AUTHOR Bach, Paul J.
TITLE Cognitive Change in Elderly Populations: "Normal" Aging, Senile Dementia and Depression.
PUB DATE May 81
NOTE 40p.: Paper presented at the Annual Convention of the Rocky Mountain Psychological Association (51st, Denver, CO, April 29-May 2, 1981).

EDRS PRICE MF01/PC02 Plus Postage.
DESCRIPTORS *Adult Development; *Aging (Individuals); *Behavior Change; *Cognitive Ability; *Depression (Psychology); Developmental Stages; Literature Reviews; *Memory; Older Adults; Psychological Patterns; Research Problems; State of the Art Reviews
IDENTIFIERS *Senile Dementia

ABSTRACT

Cognitive change in the elderly can be due to several etiological factors which are empirically difficult to separate and clinically problematic to differentiate. Normal aging is accompanied by behavioral slowing. The slowing down of psycho-motor processes results in a lowered intelligence quotient, but cannot be taken as unequivocal evidence for intellectual deterioration. Acuity of abstract non-verbal reasoning diminishes with the normal aging process, but generalizations from this evidence cannot be made without several qualifications. Dementia is a pathological process which often accompanies normal aging, is apparently physiological in origin, and is psychometrically characterized by impairment of memory and abstract verbal reasoning. Although certain post-mortem findings are associated with dementia, the severity of these physiological factors is not related to the degree of pre-mortem intellectual deterioration in a straightforward fashion. The conceptualization of dementia is unduly confused by artificial division into several subtypes. Finally, depression, a common concomitant of both the normal aging and dementing process, can result in psychomotor retardation, memory and concentration impairment, and a host of other idiosyncratic alterations of cognition. The configuration, rather than the severity of cognitive alterations, can best be used in differential diagnosis and treatment formulations, as well as in future research. (Author/NRB)

****
* Reproductions supplied by EDRS are the best that can be made *
* from the original document. *
****

ED205872

COGNITIVE CHANGE IN ELDERLY POPULATIONS: "NORMAL" AGING, SENILE DEMENTIA AND DEPRESSION

Paul J. Bach

University of Montana

U.S. DEPARTMENT OF EDUCATION
NATIONAL INSTITUTE OF EDUCATION
EDUCATIONAL RESOURCES INFORMATION CENTER (ERIC)

X This document has been reproduced as received from the person or organization originating it.

☐ Minor changes have been made to improve reproduction quality.

• Points of view or opinions stated in this document do not necessarily represent official NIE position or policy.

"PERMISSION TO REPRODUCE THIS MATERIAL HAS BEEN GRANTED BY

Paul J. Bach

TO THE EDUCATIONAL RESOURCES INFORMATION CENTER (ERIC)

CG 015364

Paper presented at

Rocky Mountain Psychological Association Convention

Denver, CO, 1981

Cognitive Changes in Elderly Populations: "Normal" Aging, Senile Dementia and Depression

The clinician faced with an elderly patient who is undergoing some detectable cognitive change is faced with a diagnostic issue of substantial proportions. Is the patient simply getting older, undergoing a dementing process or affectively disturbed? Treatment and prognosis will vary greatly on the basis of the diagnosis.

This review of the literature is undertaken to assist the clinician in the resolution of these issues. It will in turn consider empirical, conceptual and clinical issues related to the cognitive change which accompanies the normal aging process, the dementing process and depressive phenomena in the elderly. Having viewed those issues, some observations regarding necessary future research will be made.

"Normal" Aging

Empirical Issues

Physiological processes. Aging is regularly accompanied by a decrease in the number of neural cells in the cerebral cortex. This depletion can be as substantial as 45% in some areas of the brain (Brody & Vijayashankar, 1976), although it is usually less severe in most areas. Cell loss is apparently quite site-specific and not generalized (Brody & Vijayashankar, 1976) occurring, in the cortex, most profoundly in the pre-frontal and superior temporal lobe areas (Scheibel & Scheibel, 1975). Later-developing nervous tissue, that is the neo-cortex, appears more prone to cell depletion with aging than does earlier-developing cerebral structures, e.g. the limbic system (Brody & Vijayashankar, 1976). One exception is the hippocampus, which

suffers marked cell loss during aging (Scheibel & Scheibel, 1975). Quantifying the extent of cell loss is not however a highly reliable process due to the various number of laboratory techniques for quantification.

Even if a reliable method of quantification were identified, there is no direct relationship between neural depletion and either loss of cognitive function (Simon & Malamud, 1965) or chronological age. Hence cell loss cannot be adequately conceived of as the cause of cognitive change, nor as a necessary effect of aging.

Also regularly found in the aging brain is the intracellular presence of fluorescent pigments called lipochromes. Apparently some cytoplasmic function, perhaps protein synthesis and thus nurturance of the cell, is made less effective by the increase in lipochrome deposits within the neuron (Scheibel & Scheibel, 1975). Other, less regularly occurring, concomitants of aging in the brain are the decrease of extracellular space, changes in the structure of supportive neural tissue and the deposit of senile plaques (Scheibel & Scheibel, 1975). These last three changes are found primarily in the hippocampal region and do not necessarily occur in the aged brain. Their irregular occurrence may be an indication that they are pathological and not normal changes found in the aged brain (Scheibel & Scheibel, 1975).

The currently popular position in the literature is that neural degeneration is not a necessary accompaniment of the aging process and thus should be considered abnormal or pathological (Botwinick, 1980). Despite the popularity of this position, however, the above described histological changes do occur with sufficient regularity to cause some investigators (Terry & Wesniewski, 1975) to conclude on the basis of the

preponderant proportion of the evidence that neuronal loss, lipochromes and senile plaques are common in both the normally aging and the abnormally dementing brain, the difference being only quantitative.

Global intelligence. Perhaps more than any other issue related to aging, intelligence, and its apparent decline with age, has been a focus for controversy among investigators. From the perspective of the 1980's the controversy is not easy to comprehend. The conception that intelligence is not a unitary construct to be universally applied without ambiguity is very clear (Baltes, Reese, & Lipsitt, 1980), perhaps as a result of the protracted debate during the 1960's and 70's regarding the relationship between race and intelligence (Jensen, 1980). However, during the 1970's there was no small contention regarding the extent and interpretation of intellectual decline during the aging process.

The resolution of this controversy seems to revolve around a number of foci. First, everyone seems to agree that epidemiologically, various pathologies do become more frequent during old age, and that various pathologies result in intellectual decline, resulting in more frequent intellectual decline during the latter years. What one is left with, then, is the question whether non-pathological (i.e., "normal") aging also necessarily involves intellectual decline--a much more circumscribed issue (Kinsbourne, 1977).

Second, as Baltes and his colleagues have pointedly argued (Schaie, 1974; Baltes & Schaie, 1976; Baltes, Reese, & Lipsitt, 1980; Willis & Baltes, 1980) much of the evidence which has been interpreted as substantiation for the notion of universal cognitive decline in old age, was a product of methodological artifact. That is, the cross-sectional data which tends to

indicate intellectual decline over the years, does not, by the very nature of the experimental design, account for differences between the cohort groups. This is of import because consecutive generations have had varying socio-historical backgrounds and have successively obtained higher mean years of education. If one accounted for the amount of variance attributable to education and cultural experience, argues Schaie, then the apparent decline of intellectual ability with age is reduced to methodological artifact, and not real decline (Schaie, 1974). Certainly the data evaluated by this group of investigators emphasizes the contribution of generational differences to findings which imply intellectual decline.

However, as Horn and Donaldson (1976) point out, even with the effect of generational differences accounted for, the general decline of some intellectual abilities ("fluid") can be demonstrated after the age of 60, and certainly after the age of 70. Most likely there is some decrement in FSIQ after the age of 70, with some abilities deteriorating earlier or more rapidly (psychomotor abilities), others later or more slowly (verbal abilities) (Botwinick, 1970; 1980).

The intriguing facet of this controversy is that most investigators agree that there are sizable individual differences in gerontological populations, the variability of intellectual abilities being greater in this age group than in any other after the age of six years old (Schaie, 1974; Horn & Donaldson, 1976). Further, both sides of the controversy agree that, on the average, there is an inexorable decline in global intellectual ability among the elderly which begins at some time during the aging process. Finally, both

groups agree that the decline in intellectual abilities, regardless of its severity or age at onset, is not a unitary phenomenon open to simplistic interpretation.

These fundamental agreements in the midst of protracted controversy suggest to this author that the controversy has been fueled as much by socio-political considerations as by ones which are scientific.

The diversity of factors which affect intellectual ability and hence the diversity of meanings with which intellectual decline in the elderly may be embued, warrants further comment. First, it seems clear that the elderly are a heterogeneous population. Neugarten (1975) suggested that the age be typified by at least two different categories: the "young" old (55-75) and the "old" old (75+). This differentiation has become popular in the literature.

Correlations between IQ and chronological age are of the .50 magnitude (Botwinick, 1980), suggesting that age accounts for only one-quarter of the variance in intellectual ability. While this is a substantial portion of variability for which any single variable to account, it in and of itself allows only crude generalizations. Rather, even a cursory consideration of other, contributory factors to intellectual ability among the elderly would include, in addition to age: history (personal and group), education, disease process, economic status, work experience, and idiosyncratic factors (Willis & Baltes, 1980).

It is an introductory assumption, then, that intellectual decline is not a universal phenomenon, effecting the entire gerontological population in a uniform global fashion (Baltes, Reese, & Lipsitt, 1980; Horn & Donaldson, 1976).

It is possible that, were one to obtain data from very large populations of the elderly, one would find more variability of performance than in any other age group--not only in degree of impairment, but also in the pattern of various different impairments on the different dimensions evaluated (Kinsbourne, 1977). That is, not only is intellectual decline variable across elderly individuals, further there is strong evidence contraindicating an intellectual decline uniform across specific abilities within individuals. It appears to be the case that the aging process differentially impairs some intellectual abilities while leaving other abilities relatively intact. It is this differential decline, resulting in a more-or-less typical profile of abilities which can assist the clinician in differentiating between "normal" aging, dementia and depression.

However, if we agree that intellectual decline is not a uniform process among the elderly, we have not yet indicated how such decline is to be validly measured. There is not apparently an age-by-race interaction in measurement of intellectual ability (Jensen, 1980), so that problem may for the time being be laid aside. However, it is unclear the extent to which the norms against which elderly persons' scores are compared on the WAIS is adequately representative or without bias. The original Wechsler norms extend only to age 64 (Wechsler, 1955). Doppelt and Wallace (1955) extended those norms to 80 years old with a population of 475 elderly persons from the community of Kansas City. However, Eisdorfer and Cohen (1961), in obtaining equivalent norms from a North Carolina population, found that in their group raw verbal scores were higher and raw performance scales lower than the

Kansas City population--indicating regional variability among the elderly, and hence the need for national norms. Further, if the Doppelt and Wallace (1955) norms are adequate, the mean verbal IQ and mean performance IQ obtained from the norms should be equal to each other and both equal to 100. In seven studies reviewed by Price, Fein and Feiberg (1980), this was not the case. Instead, while mean VIQ was roughly 100, it was consistently larger than the mean PIQ, implying that the correction of raw performance scores in the original norm study is too small. Thus, in addition to the well-documented decline of "fluid" intelligence with aging, performance scores on the WAIS are also artificially depressed by an inadequate age correction. How this confound can be parceled-out without new norms is difficult to imagine.

Verbal-Performance ability differences. The tendency for performance scale raw scores on the WAIS to decline with age more rapidly than verbal scores (Maxwell, 1961) has been conceptualized in various ways. Schaie (1974) utilized Cattell's (1963) differentiation between "crystallized" and "fluid" intelligence to describe these differential decrements in aging. Cattell posited that crystallized intelligence was composed of those verbal abilities (semantic meaning and word fluency) which were culturally over-practiced, thus being less prone to decrement with age. "Fluid" abilities, however, (those including spatial visualization and reasoning) were said to be physiologically determined and hence more vulnerable to the aging process with its encroaching neuronal deterioration. Schaie, Rosenthal and Perlman (1953) found that while measures of fluid intelligence became lower with the age of the subject, measures of crystallized intelligence

remained unchanged or increased slightly.

Regardless of the terms used to describe the differential decrement, WAIS scale scores among geronotological populations, when factor analyzed, reveal a decrement of performance scale scores more than verbal, along with performance-learning decrements greater than in verbal-learning factors (Savage, 1977). Apparently abilities measured by the performance subscales of the WAIS are more prone to decrement with age, and the propensity to learn or re-learn new performance abilities is also decreased.

This verbal/performance difference has led some to suggest that there is differential deterioration of the right hemisphere in normal aging. Klicz (1978), in an analysis of Halstead-Reitan Neuropsychological Battery data found that tests normally associated with right hemispheric dysfunction best differentiated old from young groups. Left-hemisphere tests were equivalent across ages. Neuropsychological test performance notwithstanding, there is no histological evidence to support the notion of differential right-lateralized neuron deterioration among the "normal" aged (Bondareff, 1977). Further, there is evidence that EEG abnormalities occur most frequently among the aged in the left temporal region (Obrist & Busey, 1960). Additionally, Stern,Oster and Newport (1980) found that the perceptual-motor integration abilities on signal detection tasks, were more intact in the right hemisphere than they were in the left.

Thus it can only be noted that there are some impairments of the elderly which bear a resemblance to deficits found with right hemisphere damage, and that neuropsychological findings with left

hemisphere damaged patients are quite different from those found with the elderly. Among the elderly verbal scores are higher (Information and Vocabulary subtests of the WAIS; Botwinick, 1980). However, these similarities to lateralized injuries do not bear the inferential weight of lateralized neuronal loss among the elderly.

Memory. Alteration of mnestic abilities also regularly accompanies the normal aging process. Remote memory ("old" memories) and recognition ability are quite well preserved among the elderly, while perceptual-integrative ability and recent memory (working or intermediate recall abilities) are more impaired (Wang, 1977). The conceptualization of memory impairment in the elderly is a complex matter, however.

Short-term, or primary memory is not impaired by the aging process (Fozzard, 1980; Hartley, Harker & Walsh, 1980; Baltes, Reese & Lipsitt, 1980). That is, the elderly can hold five to seven "bits" of memory for short periods of time as adequately and accurately as can younger persons. However, when the memory is more complex or needs to be retained for a longer period of time, or needs to be remembered with interference or learned with distraction, the elderly do less well than younger groups (Botwinick, 1980).

There is substantial evidence to suggest that the elderly are more prone to forget the material if distraction occurs while the learning of the material is taking place (Baltes, Reese & Lipsitt, 1980). It may be that the elderly are more prone to errors while information is being transferred from short-term to working memory, and "divided attention" situations highlight this difficulty in a way it does not with younger persons. If such a processing decrement exists, it would

effect both the storage and retrieval of information in longer term memory (Botwinick, 1980). In the elderly, working memory (regular acquisition and retrieval of relatively complex information over intermediate periods of time) is usually deficient. Long-term memory (remote or tertiary memory) remains intact (Hartley, Harker & Walsh, 1980). In addition to distraction during learning, interference prior to recall also diminishes the accuracy of working memory more among the elderly than among younger persons (Brown, Osborn & Randt, 1981, Note 1).

All told, the evidence seems to suggest the following mechanism for memory impairment in the elderly: First, the elderly perceive and transfer to primary memory uncomplex information in a competent fashion. Distraction during this transfer does alter its accuracy somewhat more so than in younger persons, however (Botwinick, 1980). Second, transfer from primary to working memory seems most deficient, and distraction during transfer or interference prior to recall substantially impairs memory; hence a dysfunction of processing and storage. Third, as a result, retrieval and output from working memory. Fourth, however, tertiary memory of remote events, coded before the age-related processing dysfunction developed, remain intact, with adequate storage and retrieval abilities.

The type of material remembered effects memory among the elderly also. Recognition does not decline with age, while recall of verbal material is substantially impaired (Botwinick, 1980). Recall of abstract or complex material is more impaired than that of concrete or simple (Baltes, Reese & Lipsitt, 1980). It is unclear, however, whether the elderly are less <u>able</u> to recall abstract or complex

material than younger persons, or if they learn the material less rapidly. There is evidence to suggest (Huntley, Harker & Walsh, 1980; Baltes, Reese & Lipsitt, 1980) that the elderly are capable of learning as accurately material which is equally complex as younger persons if they are given sufficient time or structure. Hence the difficulty in recalling abstract, complex material may be not so much an absolute dysfunction as an impairment in speed of memorial processing.

Conceptual Issues

It has been emphasized that there is no homogeneous rate of cognitive change which accompanies normal aging. Along with chronological age, a host of other factors including education (Birren & Morrison, 1961) and idiosyncratic disease or experience greatly effect cognitive alterations among the elderly (Baltes, Reese & Lipsett, 1980).

Brain-behavior relationships. It would be conceptually most elegant to relate in rather direct fashion the physiological changes which occur in the aging brain to cognitive changes which regularly accompany aging. No such direct relationship exists. Tomlinson, Blessed and Roth (1970) found a rough quantitative relationship between the number of senile plaques in the neocortex, the number of neurofibrillary tangles in the hippocampus and the amount of granuvacular degeneration in the hippocampus with the amount of cognitive impairment exhibited in the normal elderly.

Although this comprehensive investigation indicated a relationship of the magnitude of .50 between degeneration of nervous tissue and

cognitive impairment, that is still a relationship which accounts for only 25% of the variance in cognitive change among the elderly. Further, other studies have not found the relationship to be this high (Lang, 1977).

Such variability should come as no surprise. Most neural physiological change which accompanies aging has been examined on the cellular level, whereas cognitive change is measured with psychometric instruments which estimate cognitive functions on a macroscopic level. There is error variance in both cellular and cognitive measurement. Further, cognition "occurs" not on the cellular level, but at a higher, more complex inter-cellular level at various interconnected topographical sites. This inter-cellular level is not measured at all, but simply assumed (Kinsbourne, 1977). Hence, although neuronal loss may occur, it is anybody's guess the extent to which accumulated cellular degeneration must occur at one or more topographic sites in order to be expressed as cognitive change. The neocortex is an extremely interconnected system, so that between any given neuron and another, especially in a circumscribed topographic area, there exists only a few synapses. In such an interconnected system one would expect a certain amount of redundancy and plasticity of function (Baltes, Reese & Lipsitt, 1980). Hence, even in the face of moderate depletion of neural integrity, there may well remain sufficient neural interconnection to maintain largely adequate function. Simply, substantial neural depletion does not logically lead to alteration of cognitive function.

An interesting investigation undertaken by Stein and Firl (1976)

may illustrate this notion. In an A X B design where the independent variables were age of the rat and sham versus real surgical lesions of the frontal cortex, there was an age-by-lesion interaction. Only lesioned young rats did more poorly on a previously learned task. Lesioned and sham-lesioned old rats performed equivalently, although both less well than intact young rats but better than lesioned young rats. Post-mortem histological examination of the sham-operated old rats revealed substantial age-related deterioration in the unlesioned frontal cortex, causing the investigators to suggest that the lesion in the older rats had no effect because the topographic area was already ineffective due to age. Other cortical areas in both lesioned and sham-lesioned rats apparently had imperfectly performed the function required by the experimental ta k.

Such a plasticity of function on a larger topographical scale could account for the extreme variability between cortical integrity and cognitive ability among aged persons, frustrating the search for a direct brain-behavior relationship.

Slowing. Behavioral slowing is a useful focal conceptualization of the combined effects which various cognitive alterations have upon the aging person. Simply, the aging organism perceives, assimilates and responds to stimuli less rapidly. This slowing is likely associated with a decreased rate of synaptic transmission (Jarvik, 1973). It is also likely related to efficiency of respiratory functioning, EEG slowing and evoked potential amplitude but not apparently with general health (Jarvik & Cohen, 1973).

Behavioral slowing, as it is being here considered, is not

equivalent to simply longer latency of motor response or of perceptual/sensory integration but rather to the increased length of time required to initiate and successfully complete complex or organized behavior (Birren, Woods & Williams, 1980). This slowing depresses abilities which require perceptual and cognitive integration (Botwinick, 1980), that is, those abilities often referred to as "fluid" intelligence (Lezak, 1976).

Although the primary suggestion in the literature is that this slowing is due to less rapid neural transmission (Botwinick, 1970; 1980), the suggestion has also been made (Kinsbourne 1977) that a deficit in neural inhibition, resulting behaviorally in overattention to insignificant details and uncertainty as to what constitutes an adequate solution, may also contribute to the cognitive slowing which accompanies old age.

Although it does not seem to be the case that additional time always brings performance in the aged to the level of non-aged populations (Doppelt & Wallace, 1955), nor that failure to learn new material among the elderly is simply a matter of the rate at which material is presented (Botwinick, 1970), the pacing of presented problems is less important to facilitating learning than the total amount of time allowed for the learning process to occur (Kinsbourne & Berryhill, 1972). This suggests that long inspection times, slow presentation times, and longer response times would in some instances facilitate learning in the elderly (Botwinick, 1980).

The structure of cognitive change in the elderly. Several investigators have suggested that the structure of intelligence

changes in the elderly (Botwinick, 1970; Schaie & Gribbin, 1975). Memory becomes an especially important function, combining with some verbal abilities to form a factor not found in younger populations (Price, Fein & Feiberg, 1980) although the meaning of this factor is open to dispute (Cunningham, 1980). Recall in the discussion above that memory impairment in the elderly seemed not to be in sensory, primary, or tertiary memory, but rather in the processing from primary to working memory. This would suggest that in addition to Verbal and Performance factors of intelligence in the elderly there is memory or Learning Ability factor (Savage, 1977) which apparently represents a deficit in performance learning ability more than verbal learning ability (Botwinick, 1980).

It is the suggestion of this manuscript that this is what typifies cognitive change in normally aging populations. Mnestic abilities combine with (or become more salient to) other abilities to render new learning or the application of old learning to new problems more difficult. As a result the aging person suffers a handicap relative to younger persons in the areas of flexible problem-solving, the ability to alter cognitive set and the ability to focus, sustain and re-direct attention.

However, this is an impairment qualitatively and quantitatively different from that found in dementia in that primary cognitive functions are relatively spared, and it is only these higher-order, integrative functions which are altered. One might even go so far as to suggest that the alteration of these functions is due to diffuse, minimal loss of neural efficiency, rather than more focal, more

substantial neural disorganization which occurs in dementia (Kinsbourne, 1977).

Clinical Considerations

The clinician, when evaluating the elderly patient, can benefit from being aware of certain findings which are more likely to typify the normally aging person than others. It should be reiterated at the outset that the normal aging process is a variable and heterogeneous phenomena, not easily characterized psychometrically or given to simplistic clinical description.

The WAIS along with the Halstead-Reitan Battery are probably the most regularly used clinical instruments for clinically evaluating cognitive change among the elderly, but less objective rating scales or questionnaires may also be used to obtain more qualitative information about the individual (Botwinick, 1980).

First, one would expect scores on the verbal subtests of the WAIS to be as high or higher than the scores on the performance subtests (Botwinick, 1970). This seems to be related to the relative preservation of socially-determined, crystallized intellectual abilities over more directly physiologically determined fluid intelligence. There may be some decrement in Full Scale IQ after age 70 (Botwinick, 1970; Horn & Donaldson, 1976), however the absolute level of global intelligence is not uniformly related to the overall clinical picture (Wang, 1977).

Normal aging is often accompanied by decline on measures of word fluency, psychomotor speed, inductive reasoning and spatial organization. More intact and less likely to be effected by the aging

process only are measures of number concepts and verbal meaning (Horn & Donaldson, 1976). Hence low scores on Block Design, Digit Symbol or Picture Completion late in the seventh decade or early in the eighth are a "classic aging pattern" (Gallagher, Thompson & Levy, 1980), typifying the behavioral slowing and perceptuo-motor integration deficits discussed above. Even extreme slowness may be evidence not for CNS (and hence cognitive) impairment but rather peripheral motor or sensory disease (Schaie & Schaie, 1977) requiring medical attention.

However, low scores (below age and education appropriate expectations; cf. Savage et al. 1973 for norms) on verbal subscales, especially digit span (Arenberg, 1973) or other measures of verbal learning (Savage et al. 1973) indicate a process other than normal aging.

Halstead-Reitan data is less useful quantitatively, i.e., by comparing the elderly patient's scores to norms, because non-brain-damaged elderly receive scores on these tests which are similar to younger brain-damaged patients (Gallagher, Thompson & Levy, 1980; Reed & Reitan, 1963). It is probably not useful to conceive of these two groups as cortically equivalent (senile plaques may well disrupt neurotransmission in a fashion disimilar to diffuse trauma), hence it is probably not useful to use the same norms to judge their level of neuropsychological impairment (Schaie & Schaie, 1977). Further, it is unclear the extent to which scores on neuropsychological tests are related to clinically substantial manifestations of impairment among the elderly (Price, Fein & Feiburg, 1980).

However, intra-subject performance comparisons on Halstead-Reitan test scores should also reveal greater decrement on performance of right-hemisphere related functions (spatial organization, etc.) than left hemisphere functions, i.e., crystallized intelligence (Klicz, 1978) in normally aging patients.

Regardless of the instrument, tasks which involve abstract reasoning and spatial organization will reveal more impairment than tasks which involve verbal fluency, which in turn will be more impaired than the relatively preserved abilities of verbal meaning and number operations (Willis & Baltes, 1980).

Quite simply, novelty and complexity seem to be the factors in a task which will reveal a relative decrement between the performance of the aged and younger populations, where novelty is defined as the lowered probability that the patient has previously learned to solve such a problem (Kinsbourne, 1977) and complexity is defined as the increased number of input stimuli, options to choose from, and amount of cognitive reorganization of the input to choose the correct option (Cerella & Poon, 1980). When the aging patient has difficulty with tasks which are largely attributable due to task complexity or novelty, this is likely a result of normal aging. When difficulty occurs on tasks which are neither complex nor novel, this may be a concomitant of a pathological process.

Dementia

Empirical Issues

It is estimated that 4% of the population of the United States over the age of 65 suffer from senile dementia (Merritt, 1979), and

that 10% suffer from some variant of progressive mental deterioration which nonetheless allows them continued functioning in the community.

The physiological alterations which occur in dementia are very similar to those which occur with the aging process, with the possible exception of localized and severe dendritic loss in the prefrontal and superior temporal cortex (which correlates better with psychomotor dysfunction than with chronological age) and in the hippocampus (in the very old). This dendritic loss, which isolates the cell in the neuro-communicative network and thereby alters the direction of neurotransmission, may be of more functional significance to the dementing process than actual cell body loss (Scheibel & Scheibel, 1976). Although there is a significant relationship between the degree of morphological alteration in the aging brain and the extent of mental deterioration (Roth, Tomlinson & Blessed, 1967), this relationship ranges at best between .59 and .77 and alters considerably with the specificity of identification of either variable (Jarvik, 1975).

"The first changes to be noted (in dementia) are usually a general poverty of vocabulary and range of expression. The speech becomes concrete, circumlocutory and repetitive" (Millen, 1977, p. 61). Abstract semantic categories provide more difficulties for dementing patients (Rosen, 1980) than for the non-demented elderly. Hence the alteration of verbal ability is far more profoundly affected in dementia than in normal aging (Lauter & Meyer, 1968).

Conceptual Issues

Senile dementia is only one portion (20-25%) of a larger group of organic brain syndromes commonly found in the elderly, and may be held

conceptually distinct from the others: alcoholic dementia, Korsakoff's syndrome, schizophrenia, post-traumatic encephelopathy and other, more infrequent syndromes (Sherwin & Seltzer, 1976). Like Gaul, all of senile dementia is divided into three parts: senile dementia, pre-senile dementia (Alzheimer's disease) and Pick's disease. There is no empirical reason to hold these three syndromes as etiologically distinct (Merritt, 1979; Katzman & Karasu, 1975).

Arterioschlerotic involvement and senility often occur concurrently, but are of different etiology and should be held conceptually distinct (Post, 1968; Lauter & Meyer, 1968), lest the two causes for brain degeneration be conflated and neither understood with clarity (Brody, Harman & Ordy, 1975). This is so because the degree of arterioschlerotic involvement in post-mortem findings bears no consistent relationship to the degree of ante-mortem cognitive impairment (Fisher, 1968).

At best the term dementia is only clinically descriptive of a broad range of symptoms (Wang, 1977); at worst it is too vague to be of any use (Benton, 1980). Benton (1980) proposes in the place of this broad term, four distinct clinical entities:

1. Dementia with pronounced mnestic impairment, likely as a result of bilateral temporal lobe or hippocampal lesions.

2. Dementia with agnosia, likely as a result of focal left hemisphere damage in the context of diffuse arterioschlerotic involvement.

3. Dementia with aphasia likely as a result of focal left temporal lobe damage in the context of diffuse arterioschlerotic involvement.

4. Subcortical dementia (the most common) typified by slowness of cognition, poor calculation, difficulty with abstraction.

The physiological basis for dementia is somewhat different than that of normal aging (Lauter & Meyer, 1968) although the argument can be made that the differences are insubstantial and are quantitative only (Kinsbourne, 1977). As noted above, arterioschlerotic deterioration regularly occurs along with dementia-related deterioration (Wang, 1977), making it difficult to separate the two effects.

Diffuse atrophy of cortex and to a lesser extent, ventricular dilation are the morphologically most pronounced post-mortem findings. Atrophy seems most severe in the motor and sensory association areas. Senile plaques are diffusely distributed throughout the brain--enlarged, degenerating axonal endings. Neurofibrillary tangles--the kinking of fibers within the axon which causes the neuron to expand--are also diffusely found (Merritt, 1979). Although there is no histological evidence that these degenerative phenomena tend to lateralize, there is evidence from blood-flow studies which suggest that cerebral blood flow is more significantly hampered in the left hemisphere by these phenomena than in the right (Sherwin & Seltzer, 1976). The correlation between localized senile plaques found at post-mortem and scores on a mental test battery are as high as .60 (Blessed et al. 1968), and the amount of plaque may be less severe among non-impaired aged than among the dementing elderly (Lauter & Meyer, 1968).

The effect of these more severe, likely more localized, changes in dementia is a psychomotor decrement in the step-wise motor programming

of the cortex which lies after the transition from cognitive to motor operations of the response, and can probably be best conceptualized as psychomotor degeneration (Vrtunski, Mack & Hill, 1981, Note 3).

Clinical Considerations

Identifying the elderly patient suffering from a dementing process is a more demanding nosological task than it is ordinarily conceived to be. The clinician must somehow parcel out the effects of arterioschloerotic disease (Merritt, 1979), low premorbid intelligence, psychiatric disorder, peripheral sensory disorder and acute physical illness (Kay, 1977) each of which may confound the effects of dementia on psychometric measures and clinical observations.

The dementing patient will present with symptoms of a gradual onset involving memory loss (Post, 1968), verbal knowledge, constructional ability (Sherwin & Seltzer, 1976) and diffuse impairment of attention and orientation (Botwinick, 1980).

Clinical procedures which differentiate well between dementing and non-dementing elderly are the Background Interference Procedure with the Bender-Gestalt (Carter & Straumanis, 1969), the Face-Hand Test (Fink, Green & Bender, 1952) which is also items 15-20 of the Luria-Nebraska Neuropsychological Battery (Golden, Hammeke & Purisch, 1980), and a structured mental status exam (Fink, Green & Bender, 1952). There is evidence to suggest that shorter screening batteries tend to result in more false negatives (Levy, 1980) among the elderly.

The more global is the impairment, the less likely it is that the patient is showing the signs only of old age (Gallagher, Thompson & Levy, 1980). However, because we lack age-related norms

for both competent and pathological elderly populations, much differential diagnostic testing is consigned to the realm of educated guesswork (Schaie & Schaie, 1977).

With this caveat in mind, low scores on Information, Similarities and Vocabulary of the WAIS are differentially suggestive of dementia, while low scores on Arithmetic and Picture Completion are suggestive of normal aging (Overall & Gorham, 1972). Competent performance on WAIS Verbal subscales and Halstead-Reitan Aphasia Screening Test and Speech-Sounds Test mitigate against an impression of dementia, as does competent problem-solving ability (Goldstein & Shelly, 1975).

Depression

Conceptual Issues

Severe depression and dementia are commonly found simultaneously in elderly patients (Post, 1975). Merritt (1979) estimates that one-quarter of demented patients are also depressed. Although epidemiological data are highly unreliable due to varying diagnostic procedures across countries and institutional settings, one estimate is that 4-8% of those over 65 are clinically depressed (Jarvik, 1975). Depressive features appear most prevalent during the ages 45-65 (Ciompi, 1968; Jarvik, 1975; Tomlinson et al. 1972) and decline noticeably after that time.

With this high incidence of depression and the regular admixture of depressive and dementing signs in patients, depression becomes a significant factor in the consideration of cognitive change among the elderly. It is also a perplexing differential diagnostic question, for at first glance the two sets of symptoms result in similar psychometric

findings: lowered efficiency in attention and concentration, slowness to respond, confusion, mnestic difficulties and dysfunction of abstract thinking (Miller & Lewis, 1977; Merritt, 1979).

Two interesting interactions between memory impairment and depression among the elderly are worth noting. It has been suggested that depressive features are usually seen early in the dementing process and then diminuate as the patient becomes less able to evaluate the profundity of his/her cognitive impairment. There is no relationship, however, between length of dementing disease (Kaszniak, Wilson, Lazarus, Jesson & Fox, 1981, Note 2) or degree of memory impairment and degree of depression. Second, it is sometimes assumed that depression compounds the memory impairment normally found in dementia. However, extreme and frequent complaints of memory as well as the superiority of remote over recent memory are factors more regularly found among the non-demented depressed than among brain-damaged elderly (Kahn, Zarit, Hilbert & Niederiche, 1975).

The etiological relationship between dementia and depression is not an obvious or linear one. Anterioschlerotic involvement is often found in both, but does not appear to be causally related to either (Post, 1968). Perhaps the aging process renders the insular neural cells less effective, requiring greater activity of inhibitory mechanisms (Kinsbourne, 1977). This heightened inhibition, or conversely, lowered cerebral excitability, could be expressed behaviorally as depression or cognitive concreteness (Post, 1968; Ciompi 1968). Such a possibility is intriguing, but in no way explicates a direct etiopathogenic relationship between depression and dementia.

Clinical Considerations

The absence of clear etiological differentiation between dementia and depression renders diagnostic differentiation an impressionistic undertaking at best. However, some differences can be clinically noted.

Depression most often has its onset during the ages 45-65 (Kay, 1977) while dementia occurs somewhat later. Depression ordinarily has a comparatively sudden onset (Post, 1968), marked by illness such as a cerebrovascular accident (Post, 1968) or loss of significant others or status (Libow, 1980). Depression rarely presents with any prodromal loss of cognitive efficiency (Ciompi, 1968), hence cognitive impairment followed chronologically by dysphoria is suggestive of dementia, while the opposite order suggests depression (Merritt, 1979). Decreased interests, slowing of thought and action, and dysphoria are regularly found in dementia patients who are also depressed (Kaszniak et al, 1981, Note 2) but vegetative signs (insomnia, hypochondriasis, appetitive loss, constipation) more frequently accompany uncomplicated depression (Kaszniak et al. 1981, Note 2; Libow, 1980).

Another differentiating sign to be found in the patient's history is the behavioral success at life support tasks (eating, money management, grooming): the dementing patient fails in his/her efforts, while the depressed patient often makes no effort (Kay, 1977).

Upon psychological evaluation, demented patients usually do poorly on tasks requiring attention and organization, such as Porteus Mazes, Digit Span, Rey-Osterreith Complex figure design and verbal learning tasks, while depressed patients adequately or unevenly (Winegardner,

1981, Note 4). Depressed persons will look most like demented persons on tasks requiring speed, complex activity and protracted attention or effort. When these factors are eliminated, they will appear quite undemented.

Future Research

The premier topic of investigation within the larger issue of cognitive change among the elderly has been alluded to in the above discussion of global intelligence and throughout this paper by the regular placement within quotation marks the adjective "normal" to modify "aging." Much of the literature appears to consider, and this paper has conformed to the convention, "normal aging" to be the increase in chronological age without the hindrance of pathology. Such, however, is a theoretical construct only and a rather rare empirical entity. The incidence of cardiovascular, arterioschlerotic, pulmonary and renal disease (each with its own effects on CNS functioning) as well as neurological disorder is, as a rule, significantly higher among the elderly than other age groups. The equation of "normal aging" (i.e., average, modal, typical) with aging unencumbered by disease process which effects the CNS approaches meaninglessness. The average elderly person is likely to be afflicted with a medical disorder which, however indirectly, effects the integrity of the CNS.

Thus to identify an exceptionally healthy gerontological group and demonstrate a relative absence of cognitive dysfunction proves little more than long life need not be accompanied by behavioral deficit--a fact which is unfortunately inapplicable to the vast majority of old people (Kinsbourne, 1977). If one desires to demonstrate in what way

chronological age by itself effects cognitive functioning, then one could equate groups of different ages on the basis of some measure of intellectual ability and compare their performance on a wide range of tasks. Then, after one has allowed for the fact that the groups are composed of dull younger persons or bright older persons, one can observe the effects of uncontaminated aging (Kinsbourne, 1977).

The second major research need is new norms for aged populations. Doppelt and Wallace's (1955) WAIS norms are not nationally representative (Eisdorfer & Cohen, 1961) and do not sufficiently correct raw scores on the performance subtests to render the population mean PIQ equal to 100. To compare the elderly to young adult norms with measures other than the WAIS assumes that construct, concurrent and predictive validity remains constant for a wide age range--an assumption which at least warrants empirical demonstration. Predictive validity is most affected by this assumption of constancy across chronological age because this assumption fails to recognize the very real possibility that very satisfactory and successful life is possible in elderly populations with depleted cognitive capacity. What is needed are data which differentiates test scores among the elderly which are correlated with adaptive living from test scores correlated with maladaptive and dysfunctional living. Simply, what constitutes a "low" score among the elderly compared to themselves?

Norms on the Halstead-Reitan battery and other neuropsychological measures are needed which provide cutting scores and other data which will aid in differentiating brain-damaged from non-brain-damaged

gerontological populations.

New and different memory tests designed especially for the elderly (Erickson, Poon & Walsh-Sweeney, 1980). As has been noted above, mnestic functions factor with other intellectual abilities in a different way among the elderly, and measures which differentiate this age-bound change from pathological change are needed.

Finally, information is needed which more precisely delineates the type and extent of cognitive change due to affective disorders which can effect throught processes of the depressed elderly.

This shopping list of normative data will go a long distance in conceptualizing and clinically describing the various cognitive alterations which can be found in elderly populations.

Reference Notes

1. Brown, E. R., Osborne, D. P. & Randt, C. T. Profiles of memory loss: Qualitative differences in aging. Presented at International Neuropsychological Society convention, Atlanta, 1981.

2. Kaszniak, A. W., Wilson, R. S., Lazarus, L., Jesson, J. & Fox, J. H. Memory and depression in dementia. Presented at International Neuropsychological Society convention, Atlanta, 1981.

3. Vrtunski, P. B., Mack, J. L. & Hill, G. O. Choice reaction time in senile dementia: Motor response components. Presented at International Neuropsychological Society convention, Atlanta, 1981.

4. Winegardner, J. Personal communication. 1981.

References

Arenberg, D. Cognition and aging: Verbal learning, memory, problem solving and aging. In C. Eisdorfer & M. P. Lawton (Eds.), The psychology of adult development and aging. Washington, D.C.: American Psychological Association, 1973.

Baltes, P. B., Reese, H. W. & Lipsitt, L. P. Life-span developmental psychology. In M. R. Rosenzweig & L. W. Porter (Eds.), Annual Review of Psychology, Palo Alto: Annual Reviews, 1980, 31 65-110.

Baltes, P. B. & Schaie, K. W. On the plasticity of intelligence in adulthood and old age: Where Horn and Donaldson fail. American Psychologist, 1976, 31, 720-725.

Benton, A. L. Psychological testing for brain damage. Volume 1. In H. I. Kaplan, A. M. Freedman & B. J. Sadock (Eds.), Comprehensive textbook of psychiatry/III. Baltimore: Williams & Wilkins, 1980, 966-976.

Birren, J. E. & Morrison, D. F. Analysis of the WAIS subtests in relation to age and education. Journal of Gerontology, 1961, 16 363-369.

Birren, J. E., Woods, A. M. & Williams, M. V. Behavioral slowing with age: Causes, organization and consequences. In L. W. Poon (Ed.), Aging in the 1980's: Psychological issues. Washington, D.C.: American Psychological Association, 1980.

Blessed, G., Tomlinson, B. E. & Roth, M. The association between quantitative measures of dementia and of senile change in the cerebral grey matter of elderly subjects. British Journal of Psychiatry, 1968, 114, 797-806.

Bondareff, W. The neural basis of aging. In J. E. Birren & K. W. Schaie (Eds.), Handbook of psychology of aging. New York: Van Nostrand, 1977.

Botwinick, J. Geropsychology. In P. H. Mussen & M. R. Rosenzweig (Eds.), Annual Review of Psychology. Palo Alto: Annual Reviews, 1970, 21, 239-272.

Botwinick, J. Intellectual abilities. In J. E. Birren & K. W. Schaie (Eds.), Handbook of the psychology of aging. New York: Van Nostrand, 1977.

Botwinick, J. Neuropsychology of aging. In S. B. Filskov & T. J. Boll (Eds.), Handbook of clinical neuropsychology. New York: Wiley, 1981, 135-174.

Brody, H. & Vijayashankar, N. Cell loss with aging. In K. Nandy & I. Sherwin (Eds.), The aging brain and senile dementia. New York: Plenum, 1976, 285-297.

Carter, A. & Straumanis, J. Performance of senile and healthy aged persons on the BIP Bender Test. Perceptual Motor Skills, 1969, 28, 695-698.

Cattell, R. R. Theory of fluid and crystallized intelligence: A critical experiment. Journal of Educational Psychology, 1963, 54 1-22.

Cerella, J., Poon, L. W. & Williams, D. M. Age and the complexity hypothesis. In L. W. Poon (Ed.), Aging in the 1980's: Psychological issues. Washington, D. C.: American Psychological Association, 1980.

Ciompi, L. On the relations between depression, brain damage and the aging process. In C. Mueller & L. Ciompi (Eds.), Senile dementia: Clinical and therapeutic aspects. Bern, Switzerland, 1968.

Craik, F. I. M. The nature of the age decrement in the performance on dichotic listening tasks. Quarterly Journal of Experimental Psychology, 1965, 17, 227-240.

Cunningham, W. R. Speed, age and qualitative differences in cognitive functioning. In L. W. Poon (Ed.), Aging in the 1980's: Psychological issues. Washington, D. C.: American Psychological Association, 1980.

Doppelt, J. E. & Wallace, W. L. Standardization of the WAIS for older persons. Journal of Abnormal and Social Psychology, 1955, 51, 312-330.

Eisdorfer, C. & Cohen, L. D. The generality of the WAIS standardization for the aged: A regional comparison. Journal of Abnormal and Social Psychology, 1961, 62, 520-527.

Eisdorfer, C. & Friedel, R. O. (Eds.). Cognitive and emotional disturbances in the elderly. Chicago: Year Book Medical Publishers, 1977.

Erickson, P. C., Poon, L. W. & Walsh-Sweeney, L. Clinical memory testing of the elderly. In L. W. Poon, L. Cermerk, D. Arenberg, & L. W. Thompson (Eds.),New directions in memory and aging: Proceedings of the George A. Talland Memorial Conference. Hillsdale, N.J.: Erlbaum, 1980.

Erickson, R. C. & Scott, M. L. Clinical memory testing: A review. Psychological Bulletin, 1977, 84, 1130-1149.

Fink, M., Green, T. & Bender, M. The Face-Hand Test as a diagnostic sign of organic mental syndrome. Neurology, 1952, 2, 46-58.

Fisher, C. M. Dementia in cerebrovascular disease. In R. G. Siekert & J. P. Whisnant (Eds.), Cerebral vascular disease, Sixth Conference. New York: Grume & Stratton, 1968.

Fozard, J. L. The time for remembering. In L. W. Poon (Ed.), Aging in the 1980's: Psychological issues. Washington, D. C.: American Psychological Association, 1980, 273-287.

Gallagher, D., Thompson, L. W. & Levy, S. M. Clinical psychological assessment in older adults. In L. W. Poon (Ed.), Aging in the 1980's: Psychological issues. Washington, D. C.: American Psychological Association, 1980.

Golden, C. J., Hammeke, T. A. & Purisch, A. D. The Luria-Nebraska Neuropsychological Battery Manual. Los Angeles: Western Psychological Services, 1980.

Goldstein, G. & Shelly, C. H. Similarities and differences between psychological deficit in aging and brain damage. Journal of Gerontology, 1975, 30, 448-455.

Hartley, J. T., Harker, J. O. & Walsh, D. A. Contemporary issues and new directions in adult development of learning and memory. In L. W. Poon (Ed.), Aging in the 1980's: Psychological issues. Washington, D. C.: American Psychological Association, 1980, 239-252.

Horn, J. L. & Donaldson, G. On the myth of intellectual decline in adulthood. American Psychologist, 1976, 31, 701-719.

Hoyer, W. J. & Plude, D. J. Attentional and perceptual processes in the study of cognitive aging. In L. W. Poon (Ed.), Aging in the 1980's: Psychological issues. Washington, D. C.: American Psychological Association, 1980, 227-239.

Jarvik, L. F. The aging central nervous system: Clinical aspects. In H. Brody, D. Harman & J. M. Ordy (Eds.), Clinical, morphologic and neurochemical aspects in the aging central nervous system. New York: Raven Press, 1975, 1-9.

Jarvik, L. F. & Cohen, D. A biobehavioral approach to intellectual differences in aging. In C. Eisdorfer & M. P. Lantom (Eds.), Psychology of adult development and aging. Washington, D. C.: American Psychological Association, 1973.

Jarvik, L. F., Eisdorfer, C. & Blum, J. E. (Eds.), Intellectual function in adults. New York: Springer, 1973.

Jensen, A. R. Bias in mental testing. New York: Free Press, 1980.

Kahn, R. L., Goldfarb, A. I., Pollack, M. & Peck, A. A brief ofjective measure for the determination of mental status in the aged. American Jounral of Psychiatry, 1960, 117, 326-328.

Kahn, R. L., Zarit, S. H., Hilbert, N. M. & Niederehe, G. Memory complaint and impairment in the aged. Archives of General Psychiatry, 1975, 32, 1569-1573.

Kay, D. W. K. The epidemiology and identification of brain deficit in the elderly. In C. Eisdorfer & R. O. Friedel (Eds.), Cognitive and emotional disturbances in the elderly. Chicago: Year Book Medical Publishers, 1977.

Katzman, R. & Kanasu, T. B. Differential diagnosis of dementia. In W. S. Fields (Ed.), Neurological and sensory disorders in the elderly. New York: Stratton, 1975.

Kinsbourne, M. Cognitive decline with advancing age: An interpretation. In W. L. Smith & M. Kinsbourne (Eds.), Aging and dementia. New York: Spectrum, 1977, 217-236.

Klicz, D. K. Neuropsychological evaluation in older persons. In M. Storandt, I. C. Siegler & M. F. Elias (Eds.), The clinical psychology of aging. New York: Plenum, 1978.

Lauter, H. & Meyer, J. E. Clinical and nosological concepts of senile dementia. In C. Mueller & L. Ciompi (Eds.), Senile dementia: Clinical and therapeutic aspects. Berniltans Huber, 1968.

Lezack, M. D. Neuropsychological Assessment. New York: Oxford University Press, 1976.

Levy, S. M. The psychosocial assessment of the chronically ill geriatric patient. In C. Phokopp & C. Bradley (Eds.), Medical psychology: A new perspective. New York: Academic Press, 1980.

Libow, L. S. Senile dementia and "pseudo senility": Clinical diagnosis. In C. Eisdorfer & R. O. Friedel (Eds.), Cognitive and emotional disturbances in the elderly. Chicago: Year Book Medical Publishers, 1977, 75-88.

Maxwell, A. E. Trends in cognitive ability in older age ranges. Journal of Abnormal and Social Psychology, 1961, 63, 449-452.

Merritt, H. H. Presenile and senile dementia, Alzheimer's disease and senile dementia. In H. H. Merritt (Ed.), A textbook of neurology. Sixth Ed. Philadelphia: Lea & Febiger, 1979, 484-491.

Miller, E. Abnormal aging. The psychology of senile and pre-senile dementia. New York: Wiley, 1977.

Miller, E. & Lewis, P. Recognition memory in elderly patients with depression and dementia: A signal detection analysis. Journal of Abnormal Psychology, 1977, 86, 84-86.

Neugarten, B. L. The future and the young-old. Gerontologist, 1975, 15, 4-9.

Obrist, W. P. & Busse, E. W. Temporal lobe EEG abnormalities in normal senescence. EEG and Clinical Neurophysiology, 1960, 12, 244.

Overall, J. E. & Gorham, D. R. Organicity versus old age in objective and projective test performance. Journal of Consulting and Clinical Psychology, 1972, 39, 98-105.

Post, F. The development and progress of senile dementia in relationship to the functional psychiatric disorders of later life. In C. Mueller & L. Ciompi (Eds.), Senile dementia: Clinical and therapeutic aspects. Bern: Hans Huber, 1968, 85-100.

Post, F. Dementia, depression and pseudo-dementia. In D. F. Benson & D. Blumen (Eds.), Psychiatric aspects of neurological disease. New York: Grune & Stratton, 1975.

Price, L. J., Fein, G. & Feinberg, I. Neuropsychological assessment of cognitive function in the elderly. In L. W. Poon (Ed.), Aging in the 1980's: Psychological issues. Washington, D.C.: American Psychological Association, 1980, 78-85.

Reed, H. B. C. & Reitan, R. M. A comparison of the effects of the normal aging process with the effects of organic brain damage on adaptive abilities. Journal of Gerontology, 1963, 18, 177-179.

Rosen, W. G. Verbal fluency in aging and dementia. Journal of Clinical Neuropsychology, 1980, 2, 135-146.

Savage, R. D. Intellect and personality in the aged. In W. L. Smith & M. Kinsbourne (Eds.), Aging and dementia. New York: Spectrum, 1977, 173-188.

Savage, R. D., Britton, P. G., Bolton, N. & Hall, E. H. Intellectual functioning in the aged. New York: Harper and Row, 1973.

Schaie, K. W. Translations in gerontology--From lab to life: Intellectual functioning. American Psychologist, 1974, 29, 802-815.

Schaie, K. W. & Gribbin, K. Adult development and aging. In M. R. Rosenzweig & L. W. Porter (Eds.), Annual review of psychology. Palo Alto: Annual Reviews, 1975, 26, 65-96.

Schaie, K. W., Rosenthal, F. & Perlman, R. M. Differential mental deterioration of factorially "pure" functions in later maturity. Journal of Gerontology, 1953, 8, 191-196.

Schaie, J. P. & Schaie, K. W. Psychological evaluation of the cognitively impaired elderly. In C. Eisdorfer & R. O. Friedel (Eds.), Cognitive and emotional disturbances in the elderly. Chicago: Year Book Medical Publishers, 1977.

Scheibel, M. E. & Scheibel, A. B. Structural changes in the aging brain. In H. Brody, D. Harman & J. M. Ordy (Eds.), Clinical, morphologic and neurochemical aspects in the aging central nervous system. New York: Raven Press, 1975, 10-16.

Scheibel, M. E. & Scheibel, A. B. Differential changes with aging in old and new cortices. In K. Nandy and I. Sherwin (Eds.), The aging brain and senile dementia. New York: Plenum, 1976, 39-58.

Sherwin, I. & Seltzer, B. Senile and pro-senile dementia. In K. Nandy & I. Sherwin (Eds.), The aging brain and senile dementia. New York: Plenum, 1976, 285-297.

Simon, A. & Malamud, N. Comparison of clinical and neuropathological findings in geriatric mental illness. In Psychiatric disorders of the aged, 1965, Manchester, England: Geigy, 1965.

Stein, D. G. & Firl, A. C. Brain damage and reorganization of function in old age. Experimental Neurology, 1976, 52, 157-167.

Stern, J. A., Oster, P. J. & Newport, K. Reaction time measures, hemisphoric specialization, and age. In L. W. Poon (Ed.), Aging in the 1980's: Psychological issues. Washington, D. C.: American Psychological Association, 1980, 309-327.

Terry, R. D. & Wisniewski, H. M. Structural and chemical changes of the aged human brain. In S. Gershon & A. Raskin (Eds.), Aging, Vol. 2. New York: Raven Press, 1975.

Tomlinson, B. E. Morphological brain changes in non-demented old people. In H. M. van Praag & A. F. Kalverboer (Eds.), Aging of the central nervous system. Haarlen: Bohn, 1972, 38-57.

Tomlinson, B. E., Blessed, G. & Roth, M. Observations on the brains of demented old people. Journal of Neurological Science, 1970, 11, 205-221.

Wang, H. S. Dementia of old age. In W. L. Smith & M. Kinsbourne (Eds.), Aging and dementia. New York: Spectrum, 1977, 1-24.

Wechsler, D. Manual for the Wechsler Adult Intelligence Scale. New York: Psychological Corporation, 1955.

Willis, S. L. & Baltes, P. B. Intelligence in adulthood and aging: Contemporary issues. In L. W. Poon (Ed.), Aging in the 1980's: Psychological issues. Washington, D. C.: American Psychological Association, 1980, 260-272.